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The duration of growth hormone (GH) therapy in GH deficient adults in order to improve cardiac growth, structure and function. How-ever, recent studies have raised the point that the use of GH therapy in GH deficient (GHD) adults in order to improve cardiac function is debatable.3

We have studied 51 adult patients (35 men; mean age 37±15 years; 23 childhood-onset growth hormone deficiency) with chronic GH deficient syndrome and 31 normal subjects (17 men; mean age 38±14 years). We did not find any significant differences in left ventricular mass, systolic or diastolic function indices compared with controls. By contrast, blood pressure (BP) values were lower in patients than in controls (Systolic BP: 113 (108–117) vs 135 (131–140) mmHg; Diastolic BP: 68 (59–71) vs 76 (69–83); P<0.01. Results are expressed as median and interquartile range). The duration of the exercise on the treadmill test was shorter in the GHD group (9·40 ± 6·20–10·51 s) vs 12·42 ± 12·00–14·05 s) minutes; P<0.01.4

Patients who initiated GH therapy were re-evaluated at 6, 12 and 24 months under GH therapy (11, 8 and 12 patients, respectively). No changes were observed in echocardiographic or exercise parameters after GH treatment. In view of these results we may conclude that short and long-term GH substitution in GHD syndrome is not associated with significant changes in cardiac structure or function.

Controversial data exists about the use of GH in primary heart diseases such as dilated cardiomyopathy. Previous non-randomized clinical studies using GH demonstrated an improvement in the clinical status of patients with idiopathic cardiomyopathy, whilst recent randomized placebo-controlled studies did not confirm these results.5 In the article of Cittadini et al., the authors found after only 3-month therapy in Becker and Duchene dystrophies a significant increase in left ventricular mass. An important limitation of this study, as pointed by authors in the discussion, is the small sample size treated with GH (3 patients in Duchenne group and 6 patients in Becker group) and the high prevalence of GH/IGF-1 axis abnormalities in the group treated with GH. On the other hand, therapy with recombinant human GH, commonly administered as a single weekly dose, resulting in a constant, and small elevation of circulating GH concentrations which confers a pattern completely different than pulsatile fashion. This constant elevation of GH could be similar to the situation observed in acromegalic patients, which have typically lost the pulsatile secretion. It may result in a higher rate of complications. We should not forget that the most common cause of mortality in acromegaly is cardiovascular diseases.6 Moreover, the GH-induced left ventricular hypertrophy is not usually accompanied by an improvement in left ventricular ejection fraction as it is shown in the article of Cittadini. From this evidence, a careful attitude concerning the clinical utility of GH as coadjuvant treatment in cardiomyopathies should be maintained until further data are available.

References

Growth Hormone Therapy, Is it Always Good for the Heart?

We read with interest the article of Stro¨mberg et al.1 about the effects of growth hormone (GH) therapy on cardiac structure and function in Becker and Duchenne muscular dystrophies. Large evidences demonstrate that the growth hormone/insuline-like growth factor-1 (GH/IGF-1) axis could have great relevance for the regulation of cardiac growth, structure and function.2 However, recent studies have raised the point that the use of GH therapy in GH deficient (GHD) adults in order to improve cardiac function is debatable.3

We have studied 51 adult patients (35 men; mean age 37±15 years; 23 childhood-onset growth hormone deficiency) with chronic GH deficient syndrome and 31 normal subjects (17 men; mean age 38±14 years). We did not find any significant differences in left ventricular mass, systolic or diastolic function indices compared with controls. By contrast, blood pressure (BP) values were lower in patients than in controls (Systolic BP: 113 (108–117) vs 135 (131–140) mmHg; Diastolic BP: 68 (59–71) vs 76 (69–83); P<0.01. Results are expressed as median and interquartile range). The duration of the exercise on the treadmill test was shorter in the GHD group (9·40 ± 6·20–10·51 s) vs 12·42 ± 12·00–14·05 s) minutes; P<0.01.4

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References

Verapamil, atrial fibrillation recurrences and anticoagulation

To the Editor

We read with great interest the paper by de Simone and colleagues1 showing that the addition of verapamil to class IC or III antiarrhythmic drugs significantly reduced the atrial fibrillation (AF) recurrences.

Recurrences of AF were assessed mainly on the basis of patients’ symptoms. Such a strategy may result in a failure to detect brief, symptom-free episodes of AF. Moreover, verapamil, that decreases ventricular rate during AF, could have reduced the symptoms associated with AF recurrences and the capability of patients to recognize AF. Thus, the reduction of AF recurrences with the addition of verapamil to other antiarrhythmic drugs may be strictly confined to symptomatic AF episodes. Frequent asymptomatic (and therefore unrecognized) AF recurrences may be of paramount importance as a risk factor for the occurrence of thromboembolic events.

Anticoagulant therapy is frequently stopped after sinus rhythm has been maintained for at least one month. However, effective preservation of sinus rhythm does not preclude the occurrence of cardiovascular events.2 Moreover, among the patients treated with rhythm control, morbidity and mortality may be similar whether sinus rhythm is maintained or atrial fibrillation recurred. This finding suggests that the cardiovascular risk is not reduced with rhythm control even when sinus rhythm is maintained.2 In particular, although maintaining sinus rhythm is...
generally believed to reduce the risk of stroke, patients with risk factors may have a stroke after the cessation of anticoagulant therapy, despite the maintenance of sinus rhythm.3

We agree with de Simone and colleagues that prevention of symptomatic AF recurrences by pharmacological strategies is a desirable goal. But this strategy does not preclude the occurrence of cardiovascular events in the future. The high risk for thromboembolic events associated with both, the frequent asymptomatic and unrecognized AF recurrences and persistent comorbid conditions (e.g. heart failure) may require long term anticoagulation in at least some patients with obviously 'effective' pharmacological therapy for prevention of symptomatic AF recurrences.

References


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Low cholesterol values are no longer common in China

In the editorial published in the October 2003 issue, Lewington1 stated that ‘...lower threshold such as 4 mmol/l for total cholesterol...is not uncommon in China...’ This used to be the case several decades ago but unfortunately is no longer true.2

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In China the normal plasma cholesterol values were 155 mg/dl or 4 mmol/l in 1958,3 191 mg/dl or 4.9 mmol/l in 1981,3 200 mg/dl or 5.2 mmol/l in 1997,4 and 232 mg/dl or 6.0 mmol/l in 2003.5 This alarming trend of rising ‘normal’ cholesterol values in China is a result of a change in lifestyle and dietary habits.6

Is China paying too high a price for modernization?

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